ACTA PHYSIOLOGICA SCANDINAVICA
VOL. 40. SUPPLEMENTUM 135.
FROM THE DEPARTMENT OF PHYSIOLOGY
KUNGL. VETERINÄRHÖGSKOLAN, STOCKHOLM 51.

CORTICAL RECEPTION OF TOUCH AND TASTE IN THE CAT

A Study of Single Cortical Cells

By

M. J. COHEN[1], S. LANDGREN, L. STRÖM
AND Y. ZOTTERMAN

[1]Postdoctoral Fellow of the U. S. National Science Foundation. Present address: The Biological Laboratories, Harvard University, Cambridge, Mass. U. S. A.

Introduction.

Relatively little information is available concerning the representation of sensory modalities on a cellular level in the mammalian cerebral cortex. Questions arise as to whether a single cortical cell shares afferents from more than one modality; or whether different afferent paths serving the same modality converge on the same cortical cell. The cellular basis for the differentiation of qualities within a given modality such as vision or gustation still remains obscure and the fine cortical representation of the receptive fields within any modality is largely unknown. The importance of the patterning of impulses in cortical cellular activity is also of interest. Does the discharge of impulses by a cortical cell reflect the frequency and temporal sequence seen in the peripheral neurone or is a more subtle type of coding employed?

It is the purpose of this study to attempt at least a partial solution of these questions with the hope that such information may aid in understanding the cortical elaboration of sensation.

The chorda tympani and the trigeminal component of the lingual nerve were selected as the afferent systems whose cortical representation was to be analyzed. These two nerves innervate the same general area of the tongue and share modalities such as touch and temperature in common. In addition one modality, taste, is private to the chorda tympani. The peripheral aspect of taste has been worked out in some detail in the cat (ZOTTERMAN 1935, 1936; PFAFFMANN, 1941; COHEN, HAGIWARA and ZOTTERMAN, 1955) and it was hoped that this work on the peripheral aspect would provide a basis for the study of sensory qualities at the cortical level.

It has been shown by PATTON and AMASSIAN (1952) that electrical stimulation of the chorda tympani in the cat evokes a potential which can be recorded within a localized area on the orbital surface of the cerebrum just rostral to the tip of the anterior

ectosylvian gyrus. Bilateral destruction of this region in a variety of mammals is known to cause impairment of taste (GAD, 1891; BREMER, 1923; BENJAMIN and PFAFFMANN, 1955) and it is tempting to conclude that the area within which the evoked chorda tympani cortical potential is found corresponds to the cortical projection area of the gustatory path. However, as pointed out by PATTON and AMASSIAN, the chorda tympani contains afferent fibres from taste, thermal and tactile receptors from the tongue and it cannot be assumed *a priori* that the projection area of the chorda tympani is the cortical "taste" region. Thus a further analysis of the cortical representation of tongue afferents seemed necessary using a combination of electrical and physiological stimulation to clearly define the modalities involved. This has been carried out in the present study and has led to the conclusion that the most conspicuous component of the potential evoked by the chorda tympani is due to activity in fibres responding to tactile stimulation of the tongue. In addition to "touch cells", cortical cells responding to gustatory and thermal stimuli were also investigated and provided the basis for the type of cellular analysis of sensory modalities mentioned above.

Materials and Methods.

Anaesthesia

All experiments were done on cats. A total of 38 animals were used. Nembutal, given intraperitoneally at a dosage of 40 mg/kg body weight, was used for anaesthesia when studying cortical potentials evoked by electrical stimulation of afferent nerves. The desired level of anaesthesia was maintained during the course of the experiment by intravenous injections of the same drug. Several different types of anaesthesia were tried when recording from single cortical cells stimulated by electrical or physiological means. The narcosis was usually initiated by intraperitoneal injection of Intraval (Thiopentone sodium, May and Baker) at a dosage of 40 mg/kg body weight. As the effect of this anaesthetic rapidly wears off the animal could be maintained in a stage of anaesthesia during the experiment by intravenous injection of either Intraval or 1 % chloralose. Of these two drugs, chloralose caused less depression of the cortical activity and yielded the best preparations.

In three control experiments a combination of aether during

surgical preparation and curare (d — tubocurarine) during the recording was tried but was abandoned because the resulting cortical state was more depressed than with Intraval-chloralose. In a small number of experiments the spinal cord was cut at C 1, however, this also seemed to depress cortical activity. Whether cortical depression following cord section was due to circulatory or nervous effects is not known, but it appeared that the blood supply to the hemisphere under observation was unimpaired.

Surgery

Two different approaches to the cortical projection area of the tongue were used, one direct and one indirect.

In the direct approach the hemisphere was exposed after removal of the eye, *m. temporalis*, *m. masseter* and the *os zygomaticus*. The ipsilateral mandible caudal to the molars was removed without opening the mouth cavity to expose the junction between the chorda tympani and the trigeminal component of the lingual nerve. This allowed these nerves to be cut or stimulated according to the demands of the experiment. The middle ear was opened with a dental drill and the chorda tympani exposed for recording or stimulation. In order to have the cortical hemisphere and the ipsilateral afferent nerves in the same paraffin pool, the skin was incised in a parasagittal plane just medial to the eye and ear and then ventrally towards the angulus mandibuli. The paraffin pool was kept at 37° C by a glass tube containing a heated wire coil.

The indirect approach demanded much less dissection. The skull was opened with a trephine close to the midline over the sinus frontalis to expose the cortex surrounding the sulcus cruciatus. In order to reach the tongue projection area on the orbital surface of the frontal lobe, the microelectrode was inserted at the lateral end of the sulcus cruciatus and driven ventrally through the cortex for 6 to 10 mm. This method had the advantage of a rapid dissection with little trauma to the animal and the possibility of using very light anaesthesia. The disadvantages were the inability of using electrical stimulation of the afferent nerves in combination with the physiological stimuli, and the difficulty of finding the appropriate cortical area without the aid of the evoked cortical surface potential. The indirect approach was used in one series of experiments to look for cortical cells stimulated via the tongue receptors.

Fixation of the preparation

The animal was suspended in a frame designed by Mr. G. J. WINSBURY of the department of Physiology, the Australian National University. Fixation of the skull was achieved by a technique similar to that used in the Horsley-Clarke apparatus. In experiments where ear plugs could not be used, they were replaced by a clamp fixed to the edge of the skull surrounding the exposed cortex or to the caudal process of the *os zygomaticus*. A fairly rigid fixation of the cranium was achieved this way. The greatest technical difficulty was encountered in immobilizing the cortex. The technique adopted consisted in minimizing the movement by exposing the whole hemisphere and draining the cerebrospinal fluid by opening the ventricles through the *cisterna magna* as described by LI and JASPER (1953). In addition the thorax was opened and artificial respiration given with a minimum volume of 2 % carbon dioxide in oxygen (FRANK and FUORTES 1955). Although effective, these arrangements were not enough to immobilize the cortex. Therefore a transparent plastic plate of the type described by PHILLIPS (1956) was lightly pressed against the cortical surface. The microelectrode was inserted through a hole in the plate or through the meshes of a nylon net which sometimes replaced the plate.

Recording and stimulation techniques

The microelectrodes used consisted of the conventional micropipette drawn from pyrex glass and filled with 3 M KCl. Electrodes with a tip diameter of 1 to 3 μ and a resistance less than one megohm were used for recording mass effects from pools of neurones. When recording from single cells, electrodes were used with a tip diameter less than 1 μ and a resistance of 5 to 30 megohms. The microelectrodes were inserted with a micromanipulator designed by G. J. Winsbury and described by ECCLES, FATT, LANDGREN and WINSBURY (1954).

A positivity of the recording electrode is indicated as a downward deflection in all records. The spontaneous cortical activity always present in a lightly anaesthetised preparation made it necessary to use comparatively short amplifier time constants (50 msec) when recording the slow evoked cortical potentials with the superposed sweep technique. The slight distortion of the records was of no importance for the conclusions drawn in this paper. The time course of any slow low amplitude potentials

following large potentials were always checked with long time constant (0.5 sec) records.

The cortical potentials were recorded either between a platinum wire in contact with the surface of the cortex and a reference electrode, or between the tip of a microelectrode and the same reference electrode. The reference electrode was a chlorided silver plate wrapped in gauze cloth and sewn to the inside of the scalp skin flap which formed one side of the paraffin pool. The surface electrode was a small platinum bead at the end of a platinum wire spring.

Surface potentials from the cortex and action potentials from peripheral nerves were amplified in an RC-coupled amplifier, with a time constant which could be varied from 3 to 40 msec. For microelectrode recordings a DC-amplifier with a balanced cathode follower input stage was used. This amplifier could also be connected to perform as an RC-coupled one, with a time-constant which could be varied from 5 msec to 90 sec. The potentials were displayed on two double beam CRO-s with two cameras, one for running film recording on 35 mm paper, the other for single and multiple sweep recording on stationary 35 mm film.

Two Grass stimulators (S 4) and stimulus isolation units (SIU 4) were used for electrical stimulation. They were connected through a stimulus distribution unit which permitted connection of either or both stimulators to any one of five output receptacles. The stimulus electrodes were small flexible platinum hooks and they were also used to record from the nerves.

Results.

The length of the afferent nerve paths

The chorda tympani was stimulated electrically either in the middle ear or in the lingual nerve trunk 5 to 10 mm distal to the junction of the chorda and trigeminal part of the lingual nerve. The middle ear portion of the chorda was used both for stimulation and recording; the distance from this point to the root entry of the facial nerve was 20 to 25 mm. The distance from the middle ear to the stimulation point on the chorda-lingual trunk varied from 25 to 49 mm.

The trigeminal path was stimulated either central to the junction with the chorda or in the lingual nerve 5 to 10 mm distal to the junction. The length of the peripheral conduction path was 30 mm in the latter case.

In two cats, the nerves were dissected from the tongue to where the cranial roots join the medulla in order to determine the length of the conduction paths. The following measurements were obtained.

Chorda Tympani.

Distance from the root entry of *n. facialis* to the borderline between the posterior and middle thirds of the tongue:

Exp. 21/11. 55 (cat 3.2 kg) 85 mm.
Exp. 16/2. 56 (cat 2.5 kg) 88 mm.

Distance from the root entry of *n. facialis* to the tip of the tongue:

Exp. 21/11. 55 105 mm.
Exp. 16/2. 56 118 mm.

The trigeminal component of n. lingualis

Distance from the root entry of *n. trigeminus* to the borderline between the caudal and middle thirds of the tongue:

Exp. 21/11. 55 62 mm.
Exp. 16/2. 56 70 mm.

Distance from the root entry of *n. trigeminus* to the tip of the tongue:

Exp. 21/11. 55 82 mm.
Exp. 16/2. 56 100 mm.

The length of the central pathways from the root entrances to the cerebral cortex can only be estimated roughly by measurements on dissected brains. The distance from the root entry of the facial nerve to the cortical projection area in the rostral extension of the ectosylvian gyrus is estimated as 60 mm. The corresponding distance from the root of the trigeminal nerve to the cortex is about 10 mm shorter. The total length of the chorda tympani path from the receptors of the tongue to the ipsilateral cerebral cortex is therefore approximately 140 to 180 mm and that of the trigeminal path about 110 to 150 mm.

The conduction velocity of the chorda fibres

In studying the diameters of chorda tympani fibres, ZOTTERMAN (1935) found that a few were of large size (8 μ) but the great majority of the fibres had a diameter of 4 μ. On the basis of records from single touch, cold and pain fibres in the lingual nerve of the cat ZOTTERMAN (1936) concluded that their respective relative conduction velocities were 5 : 2 : 1. The spike amplitude

of the taste and warm fibres was about 1/5 that of the largest touch fibres while the amplitude of the cold and pain spikes were 1/9 that of the touch fibres. Thus in the corda tympani or the lingual nerve, the spikes of largest amplitude were always associated with touch fibres and these fibres may be considered as the fastest of the tongue afferents.

In order to interpret the evoked cortical response, it was necessary to define the different fibre groups in the chorda tympani and to measure their thresholds and conduction velocities. The compound action potential of the chorda tympani was analyzed by differential recording from the intact chorda in the middle ear, when stimulating the lingual nerve. The potential obtained by this method is shown in Fig. 1 B. It has a rather complicated time course, but for the present purpose it is sufficient to note the small spike of shortest latency which is seen initiating the potential complex in Fig. 1 B. This spike appears with threshold stimulation of the nerve and was interpreted as due to the fastest of the chorda fibres i. e. to fibres mediating touch from the tongue.

The experimental procedure generally required that the threshold of the fastest and second fastest group of chorda fibres be determined before cutting or crushing the chorda or the lingual nerve. The conventional monophasic recording technique could thus not be adopted. In a control experiment, however, the action potential of the chorda was recorded monophasically from the lingual nerve when stimulating the chorda tympani in the middle ear. The potential is shown in Fig. 1 A and consists of two components, a spike-like potential caused by a rather synchronous group of rapidly conducting fibres and a later wave of longer duration. The initial spike potential corresponds in time and threshold to the initial spike of the differentially recorded multiphasic potential, whereas the complex of later spikes seen in the multiphasic record appear simultaneously with the later wave in the monophasic potential. The relative thresholds and the conduction velocities of the fastest (I) and second fastest (II) group of chorda fibres are given in table I.

As the efferent secretory fibres of the chorda tympani will contribute to the compound action potential of the nerve, it was considered of interest to determine the threshold and conduction velocity of this fibre group. This was done by recording from the branch of the chorda to the submaxillary gland while stimulating

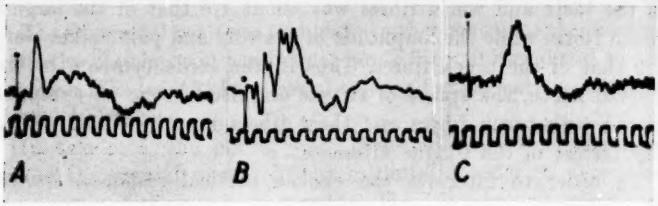

Fig. 1.
A. Monophasic spike potential recorded from the lingual nerve when stimulating the chorda tympani in the middle ear. Time: 1 msec.
B. Multiphasic action potential of chorda tympani recorded in the middle ear when stimulating the lingual nerve. Time: 1 msec.
C. Monophasic spike potential recorded from the nerve of the submaxillary gland when stimulating the chorda tympani in the middle ear. Time: 2 msec.

the chorda in the middle ear. The result is shown in Fig. 1 C. The potential was a rather uniform negative wave with a latency of 3.8 msec and a duration of 7 msec. The threshold of the potential was 1.5 times that of the lowest threshold chorda fibres recorded from the lingual nerve. The conduction velocity of the secretory fibres was 4 to 12 m/sec.

Table I

Exp.	Group I		Group II		
	Threshold	Cond. veloc. m/sec	Threshold (relative to Group I)	Cond. veloc. m/sec	
16/2. 56	1	58	1.2	35	Differential recording
1/3. 56	1	61	1.4	30	
19/3. 56	1	45	1.4	28	
10/4. 56	1	46	1.2	38	
10/4. 56	1	51	1.3	24	Monophasic recording

The records of the compound action potential of the chorda tympani thus demonstrate the existence of separable groups of afferent fibres which can be distinguished on the basis of differences in threshold and conduction velocity. The first group, with the lowest threshold and a conduction velocity of about 50 m/sec, can be assumed to correspond to the small number of large diameter touch fibres described by ZOTTERMAN. The second wave of the monophasic potential, corresponding to the second and all later components of the multiphasic potential, is apparently due to the activity of several different fibre types with conduction

velocities varying from 6—30 m/sec. Using the relative figures of ZOTTERMAN quoted above the cold fibres from the tongue would be expected to have a conduction velocity of 20 m/sec and taste and warm fibres should have a slightly higher conduction rate. These fibres should thus contribute to the initial part of the later complex of the chorda action potential, whereas the secretory fibres should give rise to the tail of this potential.

Evoked Cortical Potentials.

The evoked cortical surface potential

The cortical surface potential evoked by electrical stimulation of the ipsilateral chorda tympani is shown in Fig. 2 and consists of an initial diphasic positive-negative wave with a very constant latency and duration followed immediately by a less regular positive-negative-positive complex of low amplitude and longer duration. An initial diphasic potential with a much lower amplitude

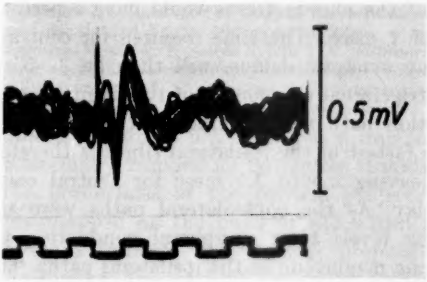

Fig. 2. Cortical surface response to single shock excitation of the ipsilateral chorda tympani. 25 superposed sweeps. Downward deflection indicates surface positivity. Time: 20 msec. Amplifier time constant TC = 40 msec.

was also obtained when the contralateral chorda was stimulated. The ipsilateral and contralateral trigeminal components of the lingual nerve also evoked similar potentials but generally of larger amplitude. These observations confirm the previous findings of PATTON and AMASSIAN (1952) and were expected from earlier investigations on other parts of the somatosensory cortex (FORBES and MORISON 1939; AMASSIAN 1953).

The latency of the initial positive phase of the evoked cortical potential (the primary response) is shown in the following table.

Table II

Afferent nerve stimulated	*Latency in msec*	
	Ipsilateral	Contralateral
Chorda tympani	4—6	5—10
Trigeminal comp. of the lingual nerve	3—4	5—6

The latencies were measured on records of 25 superposed sweeps or a series of 10 to 40 single sweeps. The ipsilateral potentials were studied in 15 and the contralateral in 6 animals.

The duration of the initial positive-negative potential was about 20 msec. The late negative wave was less regular and was sometimes difficult to separate from the spontaneous background activity. It had a latency of 20—30 msec and a duration of about 30 msec.

The peripheral conduction path of the ipsilateral chorda from the site of stimulation to the entry of the facial nerve into the medulla was about 60 mm. With a conduction velocity of 60 m/sec the fastest of the chorda fibres would have a peripheral conduction time of 1 msec. The time required for central conduction, including any synaptic delays, will then be 3—5 msec. In the case of the trigeminal component of the lingual nerve, the peripheral conduction path was 30 mm and the peripheral conduction time for the fastest of the ipsilateral fibres is therefore about 0.5 msec thus leaving 2.5 to 3.5 msec for central conduction and synaptic delay. As the contralateral paths were stimulated at corresponding levels their peripheral conduction times should be of the same magnitude as the ipsilateral paths. The latency of the contralateral responses was generally somewhat longer than the ipsilateral ones. In the case of the trigeminal path the difference in latency was 1—2 msec. The difference in latency between the contra- and ipsilateral chorda responses varied from 0 to 5 msec in different experiments. The responses from both sides were evoked by stimuli close to the threshold of the nerves and we therefore conclude that they are due to impulses in touch fibres, and that touch fibres of the chorda tympani and the trigeminal component of the lingual nerve have bilateral cortical projections.

The most conspicuous feature of the evoked surface response is the initial diphasic positive-negative potential. Experiments presented below provide further evidence that the initial positive phase of this potential when evoked by either chorda tympani

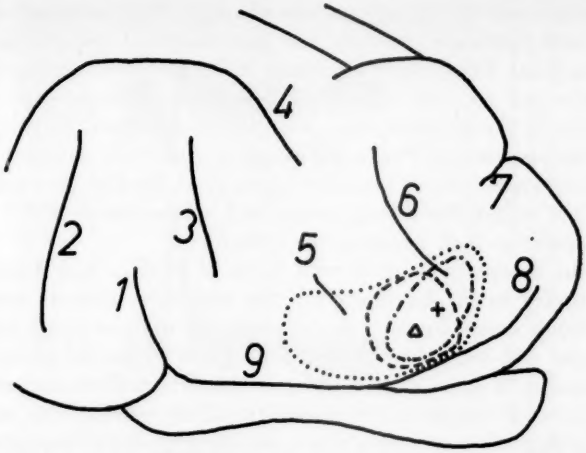

Fig. 3. Map of the cortical zones within which a cortical surface potential was evoked by electrical stimulation of the ipsilateral chorda tympani (dashed line), the ipsilateral trigeminal component of the lingual nerve (dotted line) and the contralateral trigeminal component of the lingual nerve (dash — dot line). △ maximal ipsilateral chorda and lingual-trigeminal response. + maximal contralateral lingual-trigeminal response. 1: Sulcus Sylvii; 2: S. posterior; 3: S. anterior; 4: S. suprasylvius; 5: small dimple often found anterior and ventral to the rostral ends of 3 and 4; 6: S. coronalis; 7: S. cruciatus; 8: S. praesylvius; 9: S. rhinalis. The map gives the results from one experiment.

or the trigeminal part of the lingual nerve is due to activity in the pathways normally responding to tactile stimulation of the tongue. As the borderlines of the cortical projection areas in this series were mapped on the basis of the initial positive potential, these maps indicate the cortical projection areas of touch from the tongue. The cortical projection areas of the ipsilateral chorda tympani and trigeminal component of the lingual nerve were mapped in 15 experiments and those of the contralateral nerves in 5 experiments. Fig. 3 shows the result of one such experiment. It is of interest to note the great overlap in the cortical projection areas of the different tongue afferents. In many experiments the ipsilateral chorda response area extended somewhat more ventrally than the ipsilateral projection area of the trigeminal component of the lingual nerve. The projection area of the ipsilateral or contralateral trigeminal component was always larger than the response area of the ipsilateral chorda. The contralateral chorda tympani response was comparatively weak and was seen close to the point where the ipsilateral chorda evoked a potential of maximum amplitude. In some experiments no

contralateral chorda response was obtained. The ipsilateral nerves of both pathways generally had their maximal response at the same point. The greatest amplitude of the contralateral trigeminal component response was found a few mm more rostrally. The extent of the response areas was greatly dependent on the state of the preparation. The largest response areas were obtained from lightly anaesthetized preparations in good circulatory condition with a normal body temperature and a temperature of 37° C in the paraffin bath covering the cortex.

Our observations agree with those of PATTON and AMASSIAN in regard to the localization of the responsive cortical zone. It is found dorsal to the rhinal fissure and the presylvian sulcus, caudal and ventral to the lateral tip of the coronal sulcus and cranial to a small dimple which is found rostral to the anterior sulcus and ventral to the rostral tip of the suprasylvian sulcus. It is thus within the area where the ectosylvian and suprasylvian gyri join the sigmoid gyrus.

The cortical potential evoked by stimulation of the glossopharyngeal nerve was investigated in one experiment only and the responsive zone of this nerve was found to overlap the dorsal part of the chorda-lingual area. The maximum amplitude of the glossopharyngeal response was found in the rostral part of the suprasylvian gyrus entirely outside the chorda lingual area, some 10—15 mm dorsal to the maximum response of the latter.

BREMER (1923) has described an area "profonde à la base des circonvolutions présylviennes" as a sensorimotor area for the tongue and masticatory muscles in the cat. The motor effects of direct stimulation of the cortex within the area of the evoked chorda-lingual response was therefore tested in two experiments and in both instances movements of the jaw and pharynx muscles could be elicited from this area.

The evoked potential recorded in volume at varying depths below the surface of the cortex (the focal potential)

Previous investigations (CURTIS, 1940; RENSHAW, FORBES and MORISON, 1940; BURNS and GRAFSTEIN, 1952; AMASSIAN, 1953; CRAGG, 1954) have shown that the initial portion of an evoked cortical potential changes from a positive wave to a negative wave of the same latency and duration when recording at increasing depths below the cortex surface. The same pattern was found irrespective of the function of the cortical area investigated

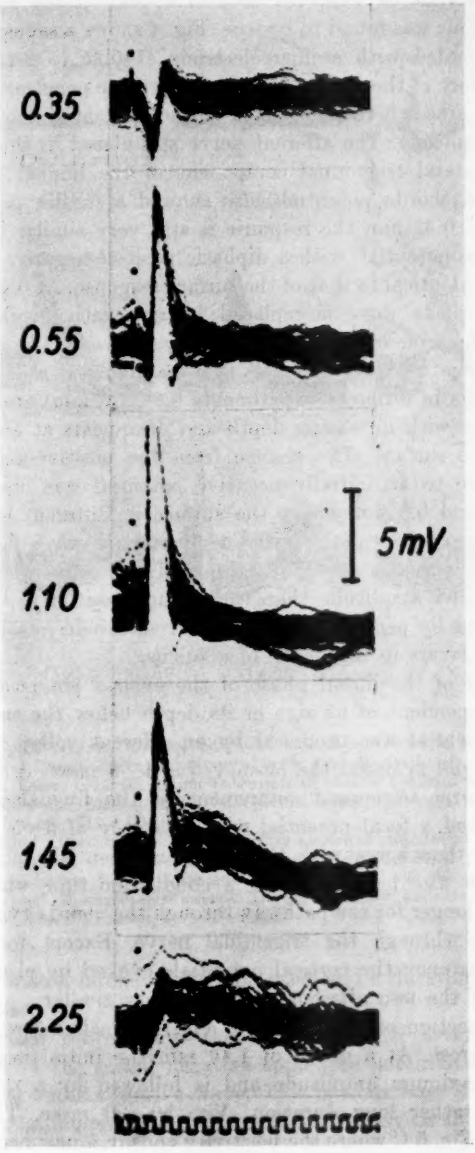

Fig. 4. The cortical focal response evoked by electrical stimulation of the ipsilateral trigeminal component of the lingual nerve and recorded with microelectrode at various depths below cortex surface. The figures indicate depth in mm below the surface. Downward deflection signals positivity in these and in all the subsequent records. Time: 5 msec. TC = 50 msec. 25 superposed sweeps.

and it should thus be expected to also appear in the cortical tongue area. This was found to be true. Fig. 4 shows a sequence of potentials recorded with a microelectrode at 0.35 to 2.25 mm below the surface of the cortex. The microelectrode penetrated the cortex in the area where the evoked surface potential showed a maximum amplitude. The afferent nerve stimulated in this case was the ipsilateral trigeminal component of the lingual nerve. The ipsilateral chorda potentials also showed a similar pattern. At a depth of 0.35 mm the response is still very similar to the evoked surface potential, with a diphasic positive-negative wave and a latency identical to that of the surface response. At 0.55 mm the initial positive wave is replaced by a negative spike-like potential of the same latency but of larger amplitude. The amplitude of this negative wave increases to a maximum at a depth of 1.10 mm (range in different experiments 0.8—1.5 mm) and then slowly declines with increasing depth and disappears at about 3 mm below the surface. The change from the positive-negative surface pattern to an initially negative potential was observed between 0.4 and 0.9 mm below the surface in different experiments. In one experiment the initial deep negative wave changed into a positive one at a depth of 2.4 mm. The positive potential showed a smaller amplitude than the surface positivity. It was possibly caused by penetration of the microelectrode into superficial cortical layers in the depth of a sulcus.

The latency of the initial phase of the evoked potential was the same independent of its sign or its depth below the surface. When the potential was produced by an afferent volley in the ipsilateral chorda tympani the latency was 4—6 msec. A volley in the ipsilateral trigeminal component of the lingual nerve, however, evoked a focal potential with a latency of 3—5 msec. The latencies thus agree with those of the potentials recorded with a surface wire lead and show a conduction time which is about 1 msec longer for the pathway through the chorda tympani than for that through the trigeminal nerve. Except for this difference in latency the cortical potentials evoked by electrical stimulation of the two afferent paths are very similar.

A close inspection of the records of Fig. 4 reveals a few other details of interest. At a depth of 1.10 mm the initial negative wave is of maximum amplitude and is followed by a shallow positivity of rather long duration. Viz.: 50—80 msec. This is better seen in Fig. 5 C where the positivity ends in a new negative

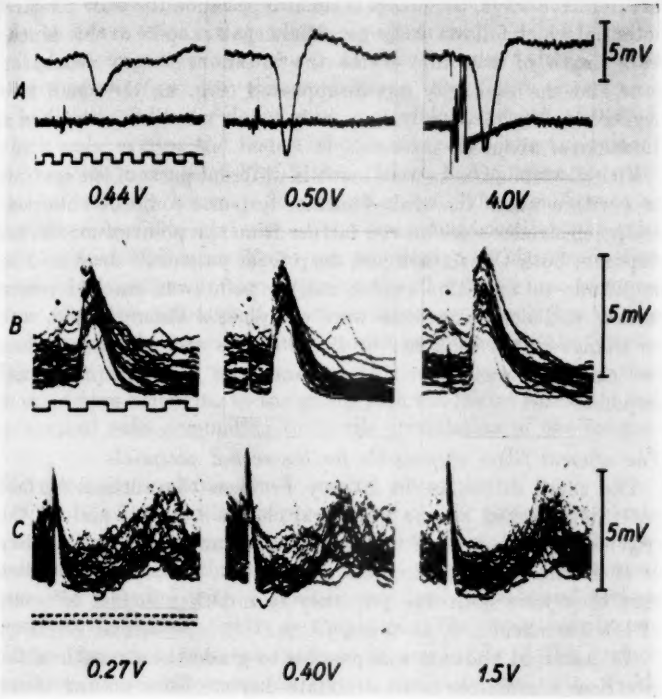

Fig. 5.

A. Upper records: Cortical potential evoked by electrical stimulation of the ipsilateral lingual nerve and recorded with microelectrode 0.1 mm below cortex surface. Lower records: Action potentials of chorda tympani fibres recorded in the middle ear. Figures under each record indicate stimulus strength in volts. Time: 5 msec. TC = 50 msec.

B. and *C*. Cortical response evoked by electrical stimulation of the ipsilateral chorda tympani and recorded with microelectrode at a depth of 1.35 mm below surface of cortex. B. and C. are similar except that C. was photographed at slower sweep speed. 25 superposed sweeps. The figures below indicate stimulus strength in volts. Time: 10 msec. TC = 50 msec.

wave with a latency of 70—80 msec and a duration of more than 80 msec.

Further down below the surface of the cortex at 1.45 mm in Fig. 4 the initial negative spike-like potential is followed by another small negative wave. This is sometimes seen as a hump on the falling phase of the large negative wave and it has a latency of 10—20 msec. It is difficult to determine the duration of this

late negative wave, because it is superimposed on the slow positive potential which follows the large initial negative spike at this depth. At a depth of 2.25 mm where the initial negativity is almost gone and the positivity has disappeared (Fig. 4), the small late negative wave has hardly been reduced in amplitude and has a duration of about 50 msec.

Microelectrode tracks were made in different parts of the cortical area within which the evoked surface response could be obtained. As the electrodes were moved farther from the point of maximum response, both the surface and deep focal potentials decreased in amplitude to the same extent until a point was reached where surface and deep responses were completely absent. There was no change in the sequence or shape of the evoked potentials as the electrode was moved away from the point of maximum response.

The afferent fibres responsible for the evoked potentials

The small difference in latency between the cortical surface potentials evoked by the ipsilateral chorda tympani and by the trigeminal component of the lingual nerve and also the similarity of their threshold to electrical stimulation raised the suspicion that they were both due primarily to impulses in fast afferents of low threshold i. e. to touch fibres.

As described above it was possible to grade the strength of the electrical stimulus so as to stimulate discrete fibre groups of the chorda tympani. The surface response as well as the focal potentials evoked by the ipsilateral chorda at various depths below the surface of the cortex were tested using different stimulus strengths. The result is shown in Fig. 5. The potentials on the left in Fig. 5 were obtained with a stimulus strength below the threshold of the second fastest group of chorda fibres, while the records in the middle column are due to a stimulus just above the threshold of this second group. The potentials in the right column of Fig. 5 are set up by a supra-maximal stimulus such that all the chorda fibres participated in the afferent volley. It is clear that the primary response, whether positive or negative in sign, is maximal in amplitude below or just above the threshold of the second fastest fibre group. With further increase of the stimulus strength the amplitude as well as the time course of the primary response remained unchanged. The latency and low threshold for the

primary cortical response indicated that it is evoked by the fast chorda fibres conducting at 30 to 60 m/sec.

The late negative wave shown in Fig. 5 C also belongs to the effects of the fast, low threshold fibres. The figure shows that it is already fully developed with a stimulus strength of 0.27 V which only excites the fastest chorda fibres. In the experiment yielding the records of Fig. 5 these fibres had a measured conduction velocity between 30 and 45 m/sec. The significance of this late negativity and also of the preceding positive potential will be discussed later. From our knowledge of the conduction rates in the different fibre types of the chorda, it now becomes clear that the initial and most conspicuous component of the evoked cortical potential is due to touch impulses from the tongue. The final proof will be given later in this paper, showing that chorda fibres belonging to the group with the lowest threshold end on cortical cells responding to tactile stimulation of the tongue.

The effect of strychnine on the evoked potentials

When a piece of filter paper soaked in 1 % strychnine nitrate solution was placed on the surface of the cortex, typical changes well known from other cortical areas occurred in the evoked potential recorded with a surface wire lead. The initial surface positivity increased slightly in amplitude. The most conspicuous change, however, was the increase in amplitude of the surface negative phase following the initial positivity (see Fig. 6 A). This large negative wave was followed immediately by one or two positive-negative waves of lower amplitude. The effect of strychnine on the evoked potential recorded by the microelectrode down to a depth of about 0.4 mm below the surface of the cortex was essentially similar to the effect on the surface response. When recording at a depth of about 1 mm where the initial phase of the focal potential is negative, local application of strychnine caused a moderate increase of the initial negative phase and a very pronounced increase of the following positivity (Fig. 6 B). In the control records this positivity was a rather shallow wave with a duration of 50—80 msec. After strychnine its amplitude exceeded that of the initial negative phase. Its duration was about 50 msec. The negative-positive complex was sometimes followed by a damped out series of negative-positive waves. When the evoked potential recorded from the surface was compared with that recorded about 1 mm below the surface it was seen that the

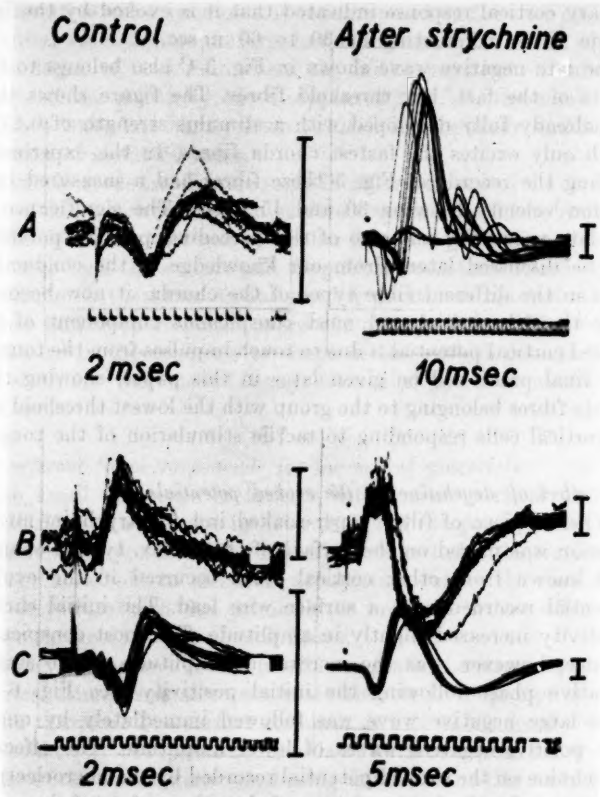

Fig. 6. Cortical response before (left) and after (right) local application of strychnine to cortex.

A. Surface response to stimulation of the ipsilateral chorda tympani. TC = 150 msec.

B. Response 1.3 mm below the surface. TC = 50 msec.

C. Surface response. TC = 40 msec. B. and C. are simultaneously recorded while stimulating the ipsilateral lingual nerve. Note the different time and voltage scales before and after strychnine application. All records formed by 10 superposed sweeps. 1 mV voltage scales to the right of each record.

large strychnine surface negative wave corresponded in time to the deep positive potential (Fig. 6 B and C).

As strychnine enhances the activity of certain neurones, the development of a large surface negative potential and a corresponding positivity below 0.8 mm may be interpreted as an

increased activity in a superficial neuron pool which sends axons or dendrites down to the cortical layers about 1 mm below the surface.

Interaction between consecutive afferent volleys

When the frequency of the shocks applied to the afferent nerve was increased above 5/sec the amplitude of the focal potential recorded at all depths below the surface of the cortex decreased. The focal potential failed almost completely at a stimulus frequency of 50/sec.

The ability of a test volley to evoke a cortical surface response at various intervals after a conditioning shock has been studied by many authors (for references see Eccles 1951). It was found that the second response was not appreciably diminished until the stimulus intervals was less than 150 msec and that at intervals less than 10 msec the test response was frequently completely abolished. In our experiments the superficial positive and the deep negative potentials were examined with conditioning and test volleys in the chorda tympani and also in the trigeminal component of the lingual nerve.

The cortical potential evoked by the test volley was reduced when the interval was shorter than 130 msec. At intervals of 20 msec the amplitude of the test response was reduced by approximately 50 %.

The effect on the focal potentials evoked by a conditioning volley in the chorda and a test volley in the trigeminal component, or vice versa, was also investigated. Where the sequence consisted of trigeminal stimulation followed by chorda stimulation, the effect on the evoked potentials was very similar to that described when test and conditioning shocks were applied to the same nerve. With the reciprocal situation, however, it was never possible to completely abolish a trigeminal evoked response no matter how closely it followed the chorda conditioning volley. We take this to indicate that there are a significant number of cortical neurones that are stimulated only by the trigeminal volley, while most of the cortical cells fired by the chorda tympani are also stimulated by the trigeminal pathway. Further evidence for the convergence of chorda and trigeminal influence on the same cortical cells will be given below in the single unit studies.

Experiments on Single Cortical Cells.

The microelectrode technique enabled us to record from single cortical cells. The type of action potential obtained when recording extracellularly close to a cell was generally a diphasic positive-negative spike with a duration of about 1 msec and an amplitude of 1 to 5 mV superimposed on a negative wave of 10 to 30 msec duration. The diphasic spike was interpreted as the propagated action potential of the neuron, and the slow negative wave as a summed effect of slow nonpropagated, post-synaptic potentials from the cell and of local as well as propagated potentials from the surrounding neurone pool. The slow negative wave is equivalent to the focal potential recorded with coarse electrodes at the corresponding depth.

When recording at a distance from a cell, monophasic negative spikes were seen and these changed to the diphasic type as the cell was approached. A microelectrode often slipped into a cell and a membrane potential of — 20 to — 70 mV was recorded together with a monophasic positive potential when the cell was fired. However, following penetration the membrane potential usually rapidly decreased and the cell fired at a high frequency and died. The observed intracellular potentials were similar to those described by BROCK, COOMBS and ECCLES (1952) for the motoneurone. A prepotential as well as a propagated spike were seen. As it was difficult to get a stable reasonably high membrane potential from these cells, very little information was gained from the intracellular records. Almost all effects described in the following part of the paper are based on extracellular records. When recording extracellularly the cells showed a constant response for 5 to more than 60 minutes and they offered an opportunity for analysis with physiological stimuli.

The convergence of chorda and trigeminal fibres on cortical cells

The first series of experiments on cortical cells made during this investigation was designed to determine if chorda and trigeminal fibres converged on single cortical cells. The ipsilateral afferent nerves were dissected free, cut and stimulated separately with electrical shocks strong enough to elicit the evoked cortical surface potential but subthreshold to the linguomandibular reflex (c. f. KING, MINZ and UNNA, 1955). It was necessary to avoid this reflex in order to prevent the preparation from moving.

As the reflex is elicited with rather weak stimulation of the trigeminal component of the lingual nerve the stimuli had to be chosen rather close to the threshold. Curare was not used in this series because it appeared to depress cortical activity. The linguo-mandibular reflex could be elicited through the ipsilateral chorda tympani but here a much stronger stimulus was required. In some experiments even a strong stimulus to the chorda did not give rise to the reflex. Unfortunately in this series of experiments the stimulus strength was not related to the group of afferent fibres stimulated, nor were the cells investigated with physiological stimuli to the tongue. Nothing is therefore known about the functions of the individual cells. However, our later experience with the difficulty of finding cells which could be excited by anything other than tactile stimulation of the tongue makes it likely that most of the cells which we found to be excited via the chorda and the trigeminal component of the lingual nerve are touch cells.

In this series a total of 80 single cortical cells were investigated. They were found between 0.2 and 3.4 mm below the surface of the cortex but more than 90% were recorded between 0.6 and 2.2 mm. Most of the cells (80 %) were spontaneously active. Of the 80 cells observed 53 could not be stimulated either via the ipsilateral chorda tympani or via the trigeminal component of the lingual nerve; 17 cells were fired by both afferent paths; 9 cells were discharged only by an afferent volley in the trigeminal component of the lingual nerve and only one cell was private to the chorda tympani path.

Fig. 7 A shows an extracellular record of a cell found at a depth of 1.25 mm below the surface of the cortex. This cell could be discharged by electrical stimulation of the ipsilateral chorda tympani as well as of the trigeminal component. The response was a diphasic positive-negative spike riding on the rising phase of a negative focal potential. The latencies from the stimulus artefact to the beginning of the negative potential was 4.6 to 5.0 msec (measured in 4 consecutive fast single sweeps similar to Fig. 7 A) and the latency of the spike potential of the cell was 5.2 to 6.4 msec. When stimulating the ipsilateral chorda tympani, on the other hand, the latency of the negative potential was 6.1 to 6.5 msec and that of the action potential of the cell was 6.4 to 7.6 msec. The response to the chorda volley thus occurred about 1 to 1.5 msec later than the trigeminal response. The

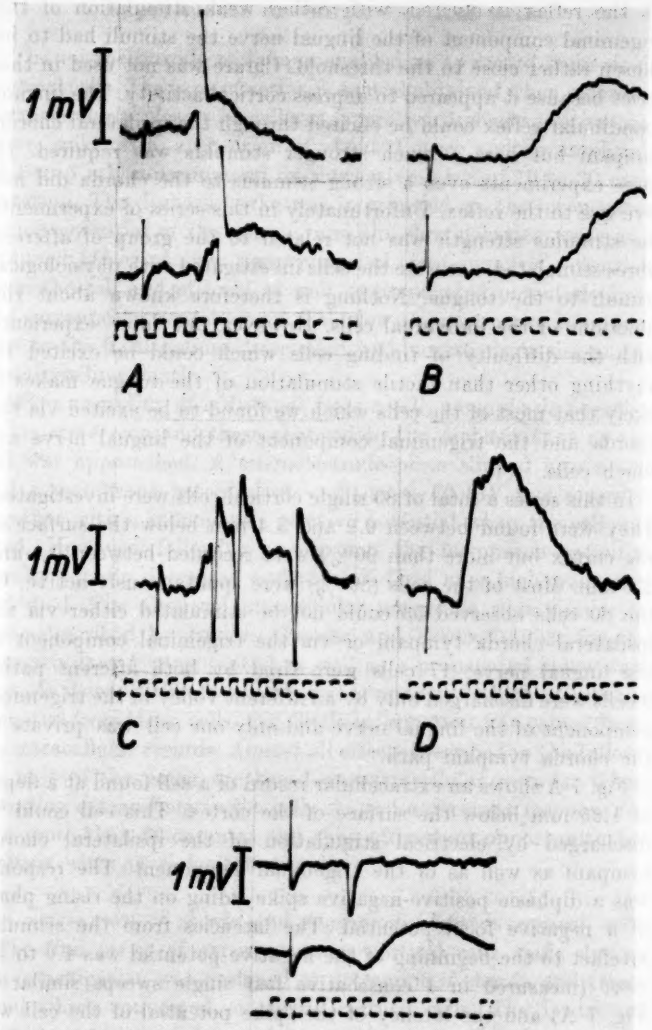

Fig. 7.

difference in latency between the propagated spikes thus agrees very closely with the corresponding difference previously described between the latencies of the evoked focal potentials. The last mentioned difference in latency, i. e. between the focal potential evoked by the chorda and by the trigeminal component of the lingual nerve is shown in Fig. 7 B. In a previous section of this paper strong evidence has been given indicating that the initial deep negativity is evoked by touch fibres in the chorda and trigeminal component. It is thus very likely that the cell shown in Fig. 7 A with a relatively short latency and fired by both afferent pathways is normally activated by tactile stimulation of the tongue (touch cell).

The important conclusions to be drawn from the above series of experiments is, however, that fibres from the chorda and the trigeminal path converge onto the same cortical cells.

In Fig. 7 C a cell is shown which was only discharged via the trigeminal path. The chorda, however, evoked a large negative potential at this point indicating that cells in the neighbourhood are effected by the chorda volley (Fig. 7 D). It can, of course, not be excluded that part of the negative chorda potential is due to a nonpropagated response in the cell which was discharged by the trigeminal volley. If this were true then it must be assumed that the convergence of chorda fibres on the cell was too small to elicit a propagated response. Evidence of such a difference in the convergence of chorda and trigeminal fibres on the same cortical cells was in fact seen in some of the cells which could be discharged via both afferent paths. In such cells a volley in the chorda gave rise to one spike while a weaker trigeminus shock elicited 2 or 3 spikes.

Fig. 7.

A. Cortical cell discharged by stimulation of ipsilat. trig. comp. of the lingual nerve (upper record) and also of ipsilat. chorda tympani (lower record). Depth 1.25 mm. Time: 2 msec. TC = 150 msec.

B. Initial negative phase of the cortical focal potential. Stimuli same as in A. Depth 1 mm. Time: 1 msec. TC = 150 msec.

C. Cortical cell discharged only by the ipsilat. trig. comp. of the lingual nerve. Depth 1 mm. Time: 1 msec. TC = 50 msec.

D. Same as C., but ipsilat. chorda stimulated. Note that cell is not fired by chorda (compare C. and D.).

E. Upper trace: Intracellular recording of a cell which is discharged only by electrical stimulation of ipsilat. chorda. Depth 2.20 mm. Time: 2 msec. TC = 50 msec. Lower trace: Surface potential recorded simultaneously with the intracellular record.

The cell which was discharged only via the chorda tympani is shown in Fig. 7 E. It was found at a depth of 2.2 mm and showed a latency of 7.3—10 msec. The long latency and the absence of a trigeminal evoked discharge suggests that this cell is not stimulated by touch fibres. In fact the depth corresponds to that of the small number of cortical cells which have been found responsive only to taste stimulation of the tongue.

It is interesting to note the large number of spontaneously active cells which could not be effected by the stimuli used. This may be due partially to the depressed state of the anaesthetized cortex. Two observations speak in favour of this suggestion. In some of these spontaneous cells in which no effect was noted when tested extracellularly with shocks to both afferent paths, a nonpropagated potential evoked by the afferent stimulation was observed when the microelectrode slipped intracellularly. The cell was thus affected by the stimulus although it was not observed in the extracellular record because the effect was too weak to elicit a propagated spike. It was also observed that the proportion of unaffected cells was smaller in very lightly anaesthetized preparations in good circulatory condition.

Touch cells

A total of 43 single cortical cells were observed which responded to touch or light pressure of the tongue. They were found at all depths between 0.5 and 2.4 mm below the surface of the cortex. Of these cells 70 % were found between 0.7 and 1.5 mm below the surface of the cortex, 20 % were found deeper and 10 % more superficially. All touch cells are thus found within the depths where the evoked potentials showed an initially negative phase and there was an obvious concentration of the observation at depths where this negative phase had its largest amplitude. When searching for cells, the microelectrode tracks were usually made within the area where the surface response had its maximal amplitude.

The stimulus used was light pressure on the surface of the tongue or a light stroke over the papillae with a glass rod. Generally a slight deformation of the tongue surface was enough to fire the cortex cell but in a few cases more pronounced pressure was necessary. Squirting a few drops of Ringer's solution on the tongue often elicited a response. In one case pricking the ipsilateral tip of the tongue with a needle was a more effective stimulus than

touching this area with the glass rod. The majority of the touch cells were tested with taste solutions (bitter, salt, sweet and sour) and with warm and cold water. No touch cell did, however, respond to these stimuli. When possible, the cells were tested with electrical stimuli to the afferent nerves.

The receptive fields

The receptor areas of the touch cells investigated were all found within the anterior two thirds of the tongue. Most of the cells (29) had an ipsilateral receptor area. Three cells showed a more diffuse receptor field located on both sides of the anterior half of the tongue. One of these cells also responded to touch within a restricted area of the *planum nasale* along both sides of the *philtrum*. Two cells showed a well localized contralateral receptor area. The borderlines of the receptive field were not adequately investigated in the remaining 9 cells. That only a small number of the cells were stimulated from the contralateral side of the tongue may be due to the fact that most of the microelectrode tracks were made within the area where the ipsilateral surface potential showed a maximum amplitude. Although the cortical projection areas overlap, the maximum of the contralateral response is found somewhat more rostrally as described previously in this paper.

When the 29 ipsilateral receptor areas were compared, it was found that 6 were located on the papillae of the dorsal surface of the tongue. Twelve were found on the ipsilateral edge but did not reach the tip of the tongue. Some of these areas were rather small and well defined. One cell responded to stimulation of the ipsilateral edge and tip and three others only to the ipsilateral tip of the tongue. Only one cell was found responsive to tactile stimulation of the ventral side of the edge of the tongue. The remaining 6 cells showed less well defined receptor fields comprising ar ge regions of the anterior half of the ipsilateral side of the tongue.

The afferent fibres exciting the touch cells

A number of cortical cells which were shown responsive to tactile stimulation of the tongue were also fired by electrical stimulation of the ipsilateral chorda tympani or the ipsilateral trigeminal component of the lingual nerve. Their threshold to electrical stimulation was measured and compared with the

threshold of action potentials recorded from the chorda tympani in the middle ear. It was found that the cortical touch cells responded to stimuli just above the threshold of the fast, low threshold chorda fibres and that this was also the threshold of the evoked cortical surface potential. These cortical cells are thus activated by the fastest of the chorda fibres with a conduction velocity of about 50 m/sec or in some cases by trigeminal fibres with approximately the same conduction velocity and threshold.

The latency of the physiologically stimulated touch cells

The latency from the moment of touching the tongue to the discharge of the cortical cell was measured in a number of experiments. The results are given in the following table.

Table III

Exp.	Number of observations	Latency, msec	
		Range	Mean
10/5. 55	2		8
24/5. 55	14	32—80	52
26/5. 55	9	11—15	12
27/6. 55	15	8—50	20
1/7. 55	48	9—34	14
23/2. 56	1		5
5/3. 56	15	11—87	32

A blunt platinum wire was used for tactile stimulation of the tongue and the contact artifact was passed through a RC amplifier and recorded on one beam of a dual beam oscilloscope to serve as a stimulus marker. In the last two experiments the impulse activity in a branch of the contralateral lingual nerve was used in order to obtain indication of the time course of the afferent volley. It is, of course, difficult to determine the exact moment of application of the touch stimulus to the receptor. The stimulating rod may have made contact with the tongue at an unknown distance from the receptor and then moved into the receptive field. If this has been the case, the latencies will be too long. As could be expected there is a great deal of variation in the measured values. Table III shows a range of latencies from 5 to 87 msec, i. e. from values comparable to the latencies found when the afferents were stimulated electrically up to values about 20 times as long as the latency for the electrically evoked response. The great variability

is in agreement with the experiments of LELE, SINCLAIR and WEDELL (1954) in which they examined the reaction time to touch in human subjects. The reaction time to touch of a finger was, as could be expected, considerably longer and varied from 180 to 300 msec depending partially on the intensity of the stimulus.

The pattern of activity of the touch cells

When a short discrete touch stimulus was applied to the tongue using a blunt rod with a diameter of about 1 mm, the cortical touch cell responded with a typical pattern of activity. On the record shown in Fig. 8 the activity of the cortical cell was recorded on one beam, and the afferent impulses in a cut branch of the contralateral lingual nerve on the other in order to monitor the form of the afferent volley. Although this index does not give an exact picture of the afferent inflow causing the cortical activity it does give an idea of the shape of the stimulus and the latency of the response. The moment of first contact with the tongue in this preparation is also marked by a small spike-like artifact visible in the lower trace of each record. With each touch of the tongue a negative wave is evoked with a duration of 20 to 30 msec and a latency of 20 msec. Two propagated action potentials are riding on top of this wave. A shallow positivity follows the negative wave and there is a pause in the impulse activity of the cell during the positive phase of the slow potential. The length of the pause was 50 to 100 msec with a mean value of 60 msec. A slow negative potential, with an irregular time course but generally of a longer duration and always with a lower amplitude than the initial negative wave, followed the positive phase and a series of propagated spikes occurred on top of the late slow negativity. The length of this late train of impulses varied greatly with the stimulus. The late response usually had a duration from 100 to 800 msec. The mean impulse frequence during the entire late series of response was 25 to 85 imp/sec. As seen in Fig. 8 B, the late series of discharges shows a certain irregular rhythm. The frequency of firing during a burst may range from 500 to 800 imp/sec and the burst may be followed by pauses in activity generally lasting between 50 and 100 msec. During the pauses, a return of the baseline to zero or a slight positivity was usually seen. It is interesting to note that the afferent volley continues during the pause in activity of the cell following the initial response

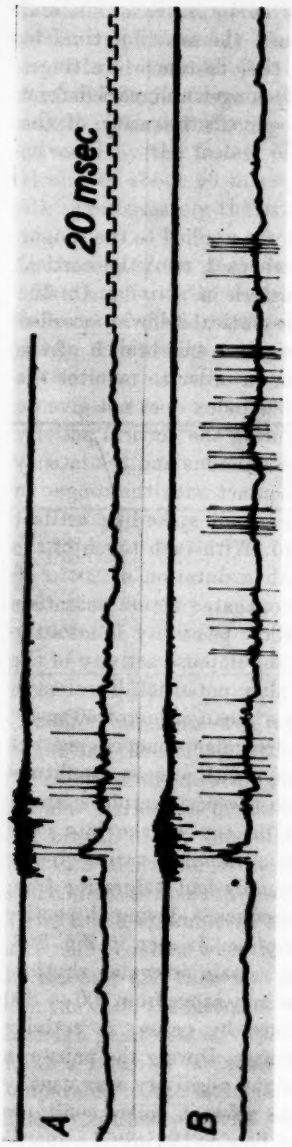

Fig. 8. *A.* and *B.* Cortical cell discharged by touching the tongue (lower trace). The afferent discharge is recorded from a cut branch of the contralateral lingual nerve to provide an indication of duration and form of the afferent volley from the tongue (upper trace). Depth 2.1 mm. TC = 50 msec.

and that the late rhythmic bursts occur in the absence of any great activity in the tongue afferents.

When the touch cell shown in Fig. 8 was electrically stimulated via the low threshold fibres of the ipsilateral chorda tympani, records of the type shown in Fig. 9 were obtained. The general pattern of activity is the same as that obtained with physiological stimulation. A slow negative wave is evoked and is seen carrying a cell action potential. This is followed by a shallow positive wave with a duration of 50 to 80 msec. No action potentials from the cell were observed during this slow positivity. The duration of the positive wave was measured in 36 records from 3 different touch cells and a mean time of 78 msec (SD $\pm$ 31 msec, 80 % of the measurements between 57 and 78 msec) was obtained. The positivity was followed by a slow irregular negative wave carrying a series of action potentials of rather high frequency. The length of this late burst of impulses

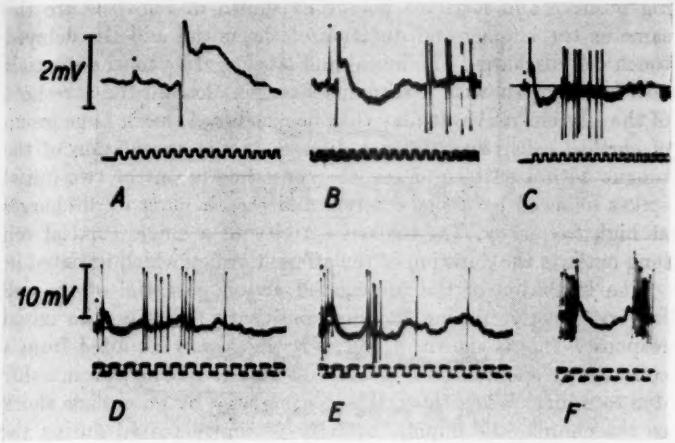

Fig. 9. Records from two cortical touch cells which are stimulated here by electrical excitation of the afferent paths.
A. — C. from a cell fired by a single electrical shock to the ipsilat. trig. comp. of the lingual nerve. Note the single spike riding on the deep initially evoked negativity followed by a slow positivity during which the cell is silent and then a burst of action potentials superposed upon a shallow negativity. Depth 1.2 mm. Time: 1 msec (A.), 5 msec (B.), 10 msec (C.).
D. — F. The response of the touch cell of Fig. 8 to a single electrical stimulation of the ipsilat. chorda. Same pattern of activity as in A. — C. Note inhibition of high frequency spontaneous activity in F. Depth 2.1 mm. Time: 20 msec. TC for all records = 50 msec.

varied greatly in successive records although stimulus strength and duration were unchanged. A certain rhythmicity (see Fig. 9 E) was seen also in the late activity induced by an electrical stimulus. It sometimes happened that cells, which usually followed the above described pattern, did not fire a spike on top of the initial negative potential although a late series of spike discharges occurred as usual, following the positive potential.

The constant pattern of activity observed in the touch cells raised the question whether this behaviour was typical for all cortical touch cells and not merely peculiar to the few cells from which we happened to record. Great similarities are noted between the time course of the single unit activity pattern and that of the slow evoked focal potentials recorded with a coarse microelectrode at a depth of 1 to 1.5 mm below the cortex surface. The latency and duration of the initial negative and the follow-

ing positive and negative potentials shown in Fig. 5 C are the same as the latency and duration of the initial and the delayed touch cell discharge. The initial and late negative focal potentials also both appear with a stimulus strength close to the threshold of the afferent nerve. It may thus be concluded that a large group of cortical cells responds to a discrete tactile stimulation of the tongue with a pattern of activity consisting of one or two initial spikes followed by a silent period and then a burst of discharges at high frequency. The evoked activity of a single cortical cell long outlasts the duration of the afferent volley which initiated it.

The inhibition of the propagated action potential of the cell is very effective during the slow positivity following the initial response. This is shown in Fig. 9 F which was recorded from a cell with a "spontaneous" activity of high frequency, presumably due to injury. When this cell was stimulated by an electric shock to the chorda, the impulse activity promptly ceased during the falling phase of the initial negative wave and did not reappear during the following positive potential. After a pause of 70 msec the cell started firing again at the previous high frequency and this activity was superposed on another slow negative wave.

The spontaneous high frequency discharge shown in Fig. 9 F developed towards the end of the period during which the cell was observed. Such a discharge is probably due to a strong local stimulation of the cell resulting from an injury to the cell membrane caused by the microelectrode. *If this assumption is true, the pause in the spontaneous discharge following the afferent volley must be due to an inhibition of the cortical cell and not to a decrease of the excitatory inflow.* The ability of the cell to fire at a very high frequency before and after the inhibition associated with the positivity shows that this inhibition is not due to refractoriness of the cell.

The effect of consecutive stimuli on the excitability of the touch cell

Some interesting information concerning the excitability of the touch cells may be obtained from experiments in which two consecutive electric shocks were applied to the afferent path at varying intervals. The records of Fig. 10 were obtained from a cell which could be activated by touching the ipsilateral edge of the tongue. A conditioning and a test shock were applied to the ipsilateral lingual nerve. At an interval between the shocks of

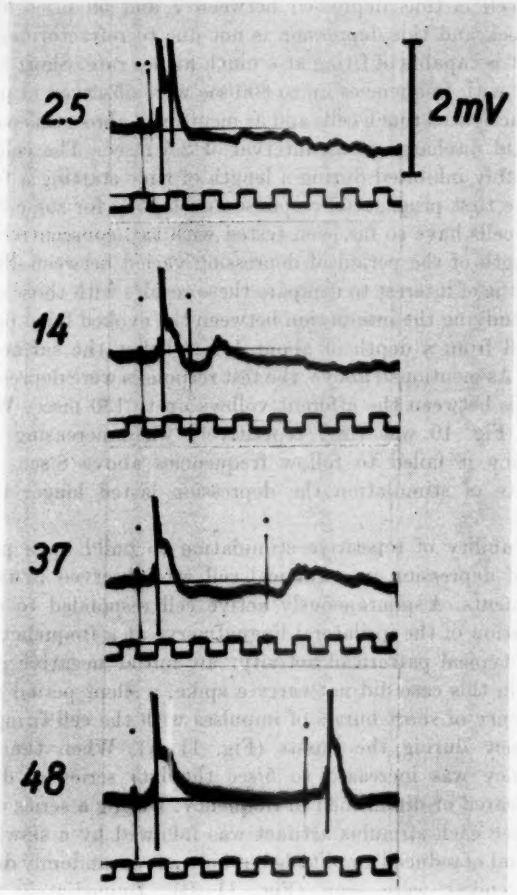

Fig. 10. Extracellular records from a cortical touch cell stimulated via the ipsilateral trigeminal path with two consecutive shocks at increasing intervals given in msec by the figures. Depth 1.4 mm. Time: 10 msec. TC = 50 msec. Stimulus artefact of conditioning shock is downward and that of the test shock is upward.

2.5 to 5 msec the cell was fired by both volleys. When the interval was 7 msec the first volley fired the cell and the second only set up a small negative potential without a propagated spike. This occurred at all intervals between 7 and 50 msec. At an interval of 50 msec the cell was again fired by both shocks. The excitability

of the cell is thus depressed between 7 and 50 msec after the first shock and this depression is not due to refractoriness of the cell as it is capable of firing at a much higher rate. Short bursts of activity with frequencies up to 800/sec were observed in physiologically activated touch cells and as mentioned above this particular cell could discharge at an interval of 2.5 msec. The cell is thus presumably inhibited during a length of time starting a few msec after the first propagated response and lasting for some 50 msec. Only 4 cells have so far been tested with two consecutive shocks. The length of the period of depression varied between 43 and 96 msec. It is of interest to compare these results with those obtained when studying the interaction between the evoked focal potentials recorded from a depth of about 1 mm below the surface of the cortex. As mentioned above, the test responses were depressed with intervals between the afferent volleys up to 130 msec. When the cell of Fig. 10 was fired repetitively with increasing stimulus frequency it failed to follow frequencies above 8/sec. Thus at this rate of stimulation the depression lasted longer than 125 msec.

The ability of repetitive stimulation to build up a prolonged state of depression in a cortical cell was observed in two other experiments. A spontaneously active cell responded to electrical stimulation of the ipsilateral lingual nerve at a frequency of 1/sec with a typical pattern of activity: an initial negative potential, which in this case did not carry a spike, a silent period and then a sequence of short bursts of impulses with the cell firing at high frequency during the bursts (Fig. 11 A). When the stimulus frequency was increased to 5/sec the late series of discharges disappeared or diminished in frequency. During a series of stimuli at 25/sec each stimulus artifact was followed by a slow negative potential of reduced amplitude but only a few, randomly discharged single spikes were seen (Fig. 11 B). Immediately after the

Fig. 11.

A. — *C.* Spontaneously active cortical cell responding to electrical stimulation of the ipsilat. trig. comp. of the lingual nerve. Stimulus artefacts marked by dots.

A. Stimulus 1/sec. Note absence of spike on evoked negative potential and the delayed bursts in which cell fires at a high frequency.

B. Stimulus 25/sec. Note decrease of spontaneous activity during stimulation and complete inhibition lasting 0.8 sec after the last stimulus.

C. Spontaneous discharge. Time: 50 c/sec.

D. and *E.* Continuous record of spontaneously active cortical cell stimulated by stroking tongue. Duration of stimulus indicated by irregularities of lower trace. Note pause in discharge following the end of stimulation. Time: 50 c/sec.

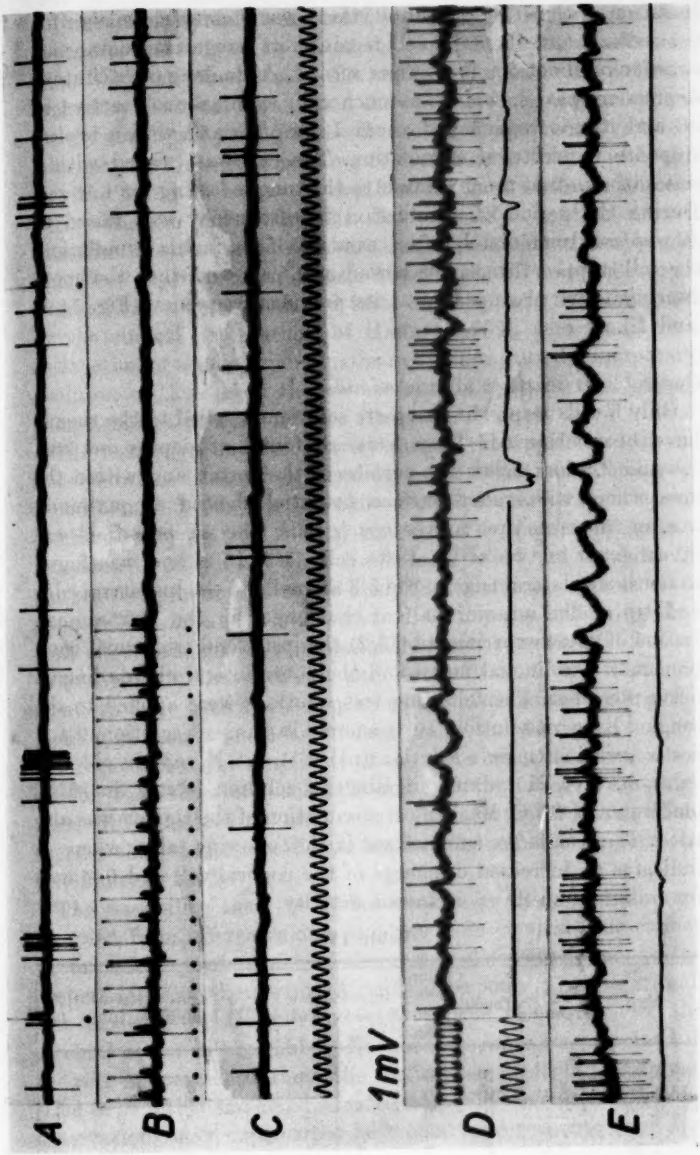

Fig. 11

cessation of the train of stimuli, there was a complete silence for 0.7 seconds and then the cell regained its irregular spontaneous activity of about 9 impulses per second. A similar postexcitatory depression was shown by a touch cell, spontaneously active at an arrhythmic frequency of about 18 imp/sec and with a typical response to electrical stimulation. The cell was excited with a series of repeated touch stimuli to the tongue lasting for 1.18 sec. During the period of stimulation the frequency was raised to 49 imp/sec. Immediately after cessation of the tactile stimulation, the cell stopped firing for a period of 0.5 sec and then the spontaneous activity reappeared at its previous frequency (Fig. 11 D and E).

Cortical cells sensitive to taste stimuli

Only 5 cells responding to taste solutions applied to the tongue have been observed. They were all found at depths between 1.8 and 2.3 mm below the surface of the cortex and within the area where the evoked surface potential showed a maximum, i. e. in the same area as the touch cells. The receptor field was investigated in two of the taste cells (5/3, 13/2) and was found to be the ipsilateral edge in the 5/3 animal and the ipsilateral edge and tip of the anterior half of the tongue in the 13/2 animal. In one of these experiments (13/2) the ipsilateral trigeminal component of the lingual nerve and the contralateral chorda-lingual nerve were cut. The following test solutions were applied to the tongue: Ringer's solution, 10 % sucrose in Ringer's solution, 0.1 M acetic acid in Ringer's solution (pH 2.5), 0.5 M sodium chloride solution, 0.02 M quinine in Ringer's solution, water at 10° C and water at 40° C. Mechanical stimulation of the tongue was also tried. The results are summarized in the following table, where + indicates an increased discharge of the cortical cell and 0 stands for no effect on its spontaneous activity.

Table IV

Exp.	Depth mm	Touch	Ringer's solution	Sucrose	Acetic acid	NaCl	Quinine	H_2O + 10°	H_2O + 40°
9/5. 55 ...		0	0			+	+	+	+
13/2. 56....	2.0	0	0		+	+	+	0	0
13/2. 56....	2.2	0	0		+	+	+	0	0
16/2. 56....	1.8	0	0		0	+	+	0	0
5/3. 56....	2.3	0	0	0	+	+	+	+	+

None of these cells responded to touch or Ringer's solution applied to the tongue. In two of the experiments touch cells were found very close to the taste cells and in still another the potential evoked by electrical stimulation showed the configuration typical for the touch cell pool. Taste cells are therefore probably closely intermixed with cells responding to touch of the tongue although they are found far below the depth where most of the touch cells were obtained and where the deep initially negative evoked potential due to tactile afferents had its maximum amplitude. This close approximation of taste and touch cells may indicate an interaction between touch and taste and this is in fact indicated by the clinical observations of HARRIS (1952), who found that destruction of the trigeminal paths in humans caused a temporary abolition of taste even though the chorda tympani and glossopharyngeal pathways for taste fibres were intact.

The change in impulse activity due to the application of the taste solutions is shown in the diagram of Fig. 12 obtained from experiment 5/3. 56. The cell showed an arrhythmic spontaneous discharge varying from 4 to 17 imp/sec. The impulse frequency increased to 30 per sec when NaCl was applied and to about 50 per sec for quinine and acetic acid. Cold water gave an increase up to about 50 and warm water to 40 imp/sec indicating that both effects are due to stimulation of water receptors (c. f. ZOTTERMAN, 1949; LILJESTRAND and ZOTTERMAN, 1954). The acid seems to have been the strongest of the taste stimuli giving the highest frequency and longest duration of the response.

The facts that the amplitude of the taste cell spikes were much smaller than those of touch cells and that the former were much more difficult to find and record from without killing them suggest that the taste cells are smaller than the touch cells (LI and JASPER, 1953).

The possibility that the spikes evoked by taste stimuli were recorded from afferent axons and not from cortical cells cannot be excluded, particularly as the taste cells were found in the deepest layer of the cortex. A cortical response to taste stimuli was recorded by GEREBTZOFF (1941) from the surface of the cerebral cortex in the rabbit. No such response was obtained in the cat. As mentioned above the cortical taste cells in this animal seem to be rather small and situated deeply in the cortex and these observations may explain the lack of a taste response from the cortical surface in the cat.

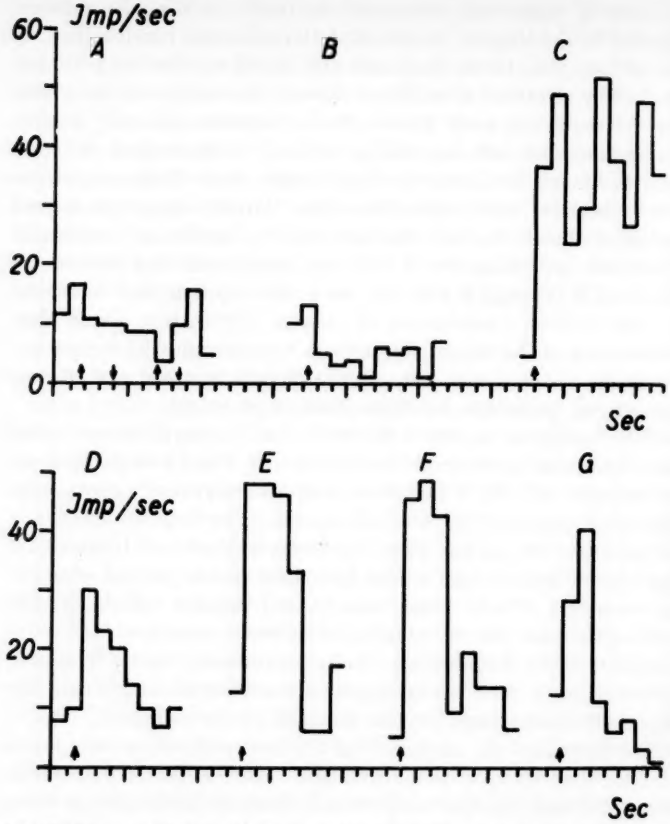

Fig. 12. Diagrams showing the impulse frequency of a cortical cell during application of taste solutions to the tongue. ↑ indicates application of the test solution. ↓ indicates end of application of the test solution.

A. Ringer's solution + 20° C.
B. 10 % Sucrose in Ringer's solution.
C. 0.1 M acetic acid in Ringer's solution, pH = 2.5.
D. 0.5 M sodium chloride solution.
E. 0.02 M quinine in Ringer's solution.
F. Water at + 10° C.
G. Water at + 40° C.

Although the experimental evidence is still meagre, some conclusions can be drawn concerning the cortical organization of taste. First, it is apparent that fibres of the gustatory pathway do project to the cortical area exited by stimulation of the ipsilateral

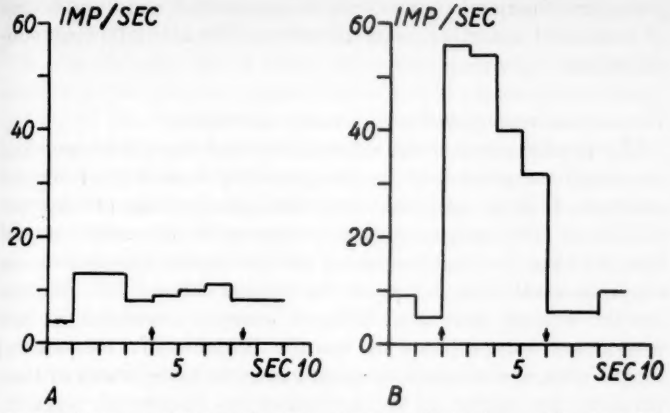

Fig. 13. Diagrams of the impulse frequency of a cortical cell responding to cooling of the tongue.

A. Application of warm water (+ 40° C) and *B*. of cold water (+ 10° C) to the tongue. The arrows indicate the beginning and end of stimulus.

chorda tympani despite the fact that the most conspicuous components of the evoked potentials are due to afferent impulses in tactile fibres. Secondly, if the response of a cortical taste cell is compared to the response of a single chorda tympani taste fibre, it is seen that the single cortical cell responds to many more types of taste stimuli than any single primary afferent taste fibre (see COHEN, HAGIWARA and ZOTTERMAN, 1955, for taste fibre response). Thus the cortical taste cell appears to integrate afferent impulses from almost all the different types of peripheral taste receptors.

Cortical cells sensitive to thermal stimuli

Two cells were found which could be stimulated by cooling the tongue but did not respond to warm water, touch or taste solutions. An exception was that one of the cells gave a weak response to 0.1 M acetic acid at body temperature. The temperature sensitive cells were both found within the touch projection area, one at a depth of 1.73 mm. The depth of the other is not known. The pattern of the response is shown in the diagrams of Fig. 13.

Discussion.

It is fully realized that data gathered from the cerebral cortex of an anaesthetized preparation may differ somewhat from the normal physiological picture. Nevertheless we feel that certain

principles of cortical activity may be approached under conditions of controlled anaesthesia and therefore offer the following considerations.

The cortical representation of sensory modalities

The topographical distribution of cortical areas corresponding to certain receptive fields in the periphery is a well established principle. It is in agreement with this principle that tactile stimulation of the tongue evokes a response in the same cortical area whether the impulses arrive via the chorda tympani or via the trigeminal component of the lingual nerve. Our findings further suggest that the different sensory modalities of the tongue are not projected to specific fields within the cortical tongue area, i. e. there is no specific area for taste, touch or temperature, but rather all the modalities are distributed together. Such an organization for the cortical representation of colours and also for skin modalities was suggested by GRANIT (1955). We have found different cells responding to either touch, taste or thermal changes of the tongue within the same microelectrode track. However, with the exception of one thermosensitive cell yielding a weak response to acid, no cell was found responding to more than one sensory modality and it thus seems possible that the modalities could be differentiated by their reception in adjacent but discrete cell pools. There may be a segregation of these cell pools in a verticle plane at different levels below the cortical surface as suggested by the fact that all the taste cells were found at a depth of about 2 mm while most touch cells were recorded at 0.7—1.5 mm.

In regard to receptive fields, GRANIT writes in his Silliman lectures (1955): "I would not be surprised if the different central receivers were correspondingly organized, some to take care of large receptive fields and others of small ones." The present experiments have offered examples of such receivers. Cortical touch cells occur with large receptive fields covering both sides of the tongue and in one case not only the tongue but also a part of the nose. Other cortical touch cells have a receptive field restricted to a distinct area a few mm in diameter on the ipsilateral dorsal surface of the tongue. It may be that certain of the cortical cells with a large receptive field simply serve to denote the general area (tongue) and the specific modality (touch) involved. Other

cortical cells, responding to a more localized receptive field could provide a "local sign" for the stimulus.

It was thought that a study of cortical potentials evoked by activity in the chorda tympani nerve would enable definite localization of the cortical taste area. It was found, however, that at least the initial and most conspicuous component of the potential evoked by ipsilateral and contralateral stimulation of the chorda was due to activity in pathways responding to tactile stimulation. The small late negative wave appearing as a hump on the falling phase of the deep negative focal potential may be a sign of gustatory activity but this remains to be verified. It is clear from single unit studies that the gustatory path does reach the cortical projection area of the chorda tympani but nothing can be said of the precise extent of the gustatory projection area.

The relationship between spontaneous and evoked cortical activity

The problem concerning the relationship between the slow spontaneous brain waves, the slow evoked cortical potentials and the activity of individual cortical cells is of great importance for the interpretation of the EEG. An observation often made during this study may throw some light on the correlation between the two first mentioned types of brain waves. It was found that the fluctuations of the baseline due to slow spontaneous cortical potentials decreased during and immediately after the primary response evoked by a strong afferent volley (Fig. 5 B). Thus the spontaneous waves invade less easily or do not originate in a cortical area during and just after the reception of an afferent volley. This suggests that the slow spontaneous potentials and the primary evoked response are due to activity in the same cortical structure.

In our records from single cortical touch cells there was a definite relationship between slow evoked potentials and propagated cell spikes. The spike discharge always appeared simultaneously with slow negative potentials and generally no spike discharge was seen during the slow positive potential following firing of the unit.

Interpretation of evoked slow potentials

The similarity between the extracellular records of Fig. 9. and the intracellular ones obtained by BROCK, COOMBS and ECCLES (1952) from single motoneurones in the spinal cord and by EYZAGUIRRE and KUFFLER (1955 a) from the stretch receptor neurone

of the crayfish, suggests that the slow negative waves are the summed result of synchronous excitatory postsynaptic potentials or dendritic generator potentials set up by the afferent volley in the touch cell pool. In fact the primary negative phase of the focal potential, which carries the initial burst of spike discharge, has generally been interpreted as a postsynaptic potential set up in the apical dendrites by the afferent volley (CHANG and KAADA, 1950, ECCLES, 1951, LI and JASPER, 1953). The true time course of this slow negative potential is difficult to assess as it seems to be cut short by a potential of opposite direction. It is, however, of a type similar to that of the excitatory postsynaptic potential of the motoneurone. The late deep negative wave carrying the delayed series of touch cell discharges seems to be built up by summation of smaller waves and reminds one of the long lasting generator potentials of the stretch receptor neurone of Crustacea (EYZAGUIRRE and KUFFLER, 1955 a).

It is also tempting to interpret the slow positive potential which always coincides with the pause in the propagated discharge as the expression of an inhibitory process analogous to the inhibitory process in the motoneurone, the crustacean muscle or the crustacean stretch receptor cell (BROCK, COOMBS and ECCLES, 1952; FATT and KATZ, 1953; EYZAGUIRRE and KUFFLER, 1955 b). The final proof must, however, await intracellular recording.

In this connection it is of interest to note the observation of TASAKI, POLLEY and ORREGO (1954) that an electric shock to the radiation fibres could give rise to a hyperpolarization of cells in the striate cortex. The "negative swing", which was recorded intracellularly, was able to stop a spontaneous injury discharge and appeared with a latency of 2—2.5 msec. Intracellular hyperpolarization of cortical cells appearing with a latency compatible with our extracellularly recorded positive wave and with the simultaneous inhibition of the spike discharge was also observed by ALBE-FESSARD and BUSER (1955).

Our observation that the surface negative potential following the initial response changes into a positive wave of similar time course (Fig. 6 B and C) with increasing depth below the cortical surface, confirms the findings of LI, CULLEN and JASPER (1956). These authors described a similar turn over of a late negative potential recorded from the superficial layers of the somatosensory cortex in response to stimulation of the thalamic relay nucleus. They interpret the change of the positive-negative surface potential

into a negative-positive deep potential as evidence of conduction of impulses along the apical dendrites from the fourth cortical layers towards the surface. However, the fact that local strychnine application enhances the later components of the evoked potentials far more than the initial response, indicates that relayed interneuronal activity makes a major contribution to the surface negative and deep positive potentials. We are therefore inclined to interpret the negative superficial potential and its corresponding deep positivity as elicited in a pool of neurones superficial to the touch cells and with processes projecting down to the latter.

CHANG (1955) described the evoked potentials recorded from the surface of the motor cortex when stimulating the pyramidal tract. His late surface negative potential (component 4) was shown to be generated by interneurones activated via axon collaterals of the large pyramidal cells. Our late surface negative potential corresponds in time to Chang's component 4 and may therefore be of similar origin, *i. e.* generated by a superficial interneurone pool activated secondarily by the primary cortical touch cells.

The discharge pattern in single cortical cells

The pause in the touch cell discharge following the initial impulses is a very interesting feature independent of the role played by the slow positive potential in its creation. It is apparent that the pause is not due to a lack of recovery of the cell after its previous discharge as the cell is readily discharged at intervals of less than 2 msec. The fact that an injury discharge can be stopped for a definite period after the injured cell has received an afferent volley indicates that the inhibition must be acting on the cell itself (Fig. 9 F). Thus the inhibition in these cortical cells appears to be an active process exerted directly on the cell and not merely due to a lack of stimulation or to refractoriness in the cell itself or in paths playing upon the cell.

Cells in the cat's cerebral cortex showing a pattern of activity similar to that of the touch cells of Figs. 8—10 have been repeatedly described in the recent literature. Thus AMASSIAN (1953) described cells found in the somatosensory cortex which respond to electrical stimulation of the ulnar nerve with an initial burst of impulses, a silent period and then a delayed discharge. The length of the silent period was about 100 msec. Similar observations were made by LI, CULLEN and JASPER (1956) also on records from neurones in the somatosensory cortex and by CREUTZFELDT,

BAUMGARTNER and SCHOEN (1956) from cells found in the motor and in the visual cortex.

The excitability curve of a single cortical sensory neurone was drawn by LI et al. on the basis of experiments with conditioning and test shocks applied to the thalamic relay nucleus. This curve shows an initial hyperexcitability lasting for 5—10 msec after the arrival of the afferent volley. During the following 30—40 msec the cortical cell was depressed and thereafter a new period of hyperexcitability lasting for 100—150 msec appeared. The pattern of activity of the cortical touch cell discharge described in this series of experiments agrees well with the excitability curve of LI et al. and so do the results of our excitability tests as shown in Fig. 10.

Still more information concerning a delayed period of depression of cortical cells may be obtained from previous investigations. Thus CHANG (1950, 1951) showed that the neurones of the auditory cortex were depressed during a period of 50 msec immediately following the primary response to a sound stimulus. Studying the cortical potential evoked by an antidromic volley in the axons of Betz cells, CHANG (1955) obtained evidence for a depression of excitability in the pyramidal cell. The depression was caused by two different processes, one of short duration corresponding to an absolute and relative refractory period of the cell and another starting with a latency of 10 msec and lasting for 60—100 msec. The later depression corresponds well in time to the period of inhibition of the touch cell. CLARE and BISHOP (1955) have also provided evidence for a depression of neurones of the visual cortex following an initial facilitation.

It should finally be mentioned that TASAKI, POLLEY and ORREGO (1954) in cells of the lateral geniculate body found a similar silent period following the response to an afferent volley in the optic nerve.

The above reviewed results show that a pattern of activity consisting of a short discharge and a silent period followed by a delayed burst of activity is typical of certain cortical neurones found in widely different parts of the cerebral cortex and also in lower integrating regions such as the lateral geniculate body. The question then arises whether the described pattern may be important in certain unknown functions of integrating neurones and whether the cortical neurones showing this pattern may belong to a functionally related group of cells appearing in the

sensory as well as in the motor cortex. Some suggestions may be offered in response to the last of these questions.

In the case of the cortical cells responding to tactile stimuli from the tongue, two conclusions may be drawn. One is that these cells belong to the largest of the cortical neurones as judged from their large extracellularly recorded spike amplitude (c. f. LI and JASPER, 1953). They are most commonly found at depths between 0.7 and 1.5 mm below the cortical surface and thus probably belong to the group of large pyramidal cells of layers III and V.

It may secondly be concluded that the touch cells belong to the groups of cortical neurones which are first thrown into action by the afferent volley because their initial discharge is found on the rising phase of the primary cortical response. The observations of CHANG referred to above indicate that the large pyramidal cells of the motor cortex may show a similar pattern. In regard to other observations made on single cortical cells using microelectrode recording it may be assumed that the technique would favour a selection of records from large cortical cells. It can therefore be suggested that the pattern of discharge shown by the cortical touch cell may be of importance for some function common to a certain group of large pyramidal cells appearing in functionally different areas of the cerebral cortex.

The following hypothesis is offered as a possible explanation of the functional role played by this particular cellular discharge pattern. The initial discharge, consisting of only one or two spikes, is assumed to start the cerebral elaboration of incoming information, *i. e.*, to activate distant cortical and thalamic neurone pools. Its brief nature may be of importance in preventing the penetration of the input signal into the major efferent channels. The cerebral treatment is assumed to be occurring during the period of inhibition and consists of an activation of interneurone pools in the same cortical region as well as in the distant cortical association areas. The thalamic nuclei involved in cortico-thalamo-cortical reverberations would also be thrown into play at this time (c. f. JASPER and AJMONE-MARSAN, 1950). The discharge of these distant cell pools is assumed to be reflected back upon the touch cell as excitatory or inhibitory impulses which reach final integration in the dendritic membrane of the touch cell. The delayed high frequency discharge following the period of inhibition may then be interpreted as the final result of the cerebral treat-

ment. Because of its duration and high frequency, the terminal burst of impulses may be capable of penetrating through long chains of synapses and thereby force its way out into the efferent paths. The period of inhibition may thus be of importance in cutting short the initial discharge so that it penetrates only into the analyzing paths and not into the major efferent outflow channels. It would also prevent later afferent signals from disturbing the cortical elaboration under progress and lastly it may serve to keep the cortical cell in a state of preparedness for the integration of the reverberating activity.

Cortical inhibition

Several mechanisms may be postulated for the inhibition following the initial discharge of the cortical touch cell. A most attractive analogy seems to be the Renshaw cell inhibition of the spinal motoneurones (ECCLES, FATT and KOKETSU, 1954). This self-inhibitory mechanism implies that impulses in collaterals of the motoneurone axon excite interneurones (Renshaw cells) which return a series of inhibitory impulses to the motoneurone. On the basis of this analogy the inhibition of the cortical touch cell should be due to a negative feed back mechanism utilizing axon collaterals and short intracortical interneurone chains. CHANG (1955) has demonstrated the existence of self-excitation in the large pyramidal cells in the motor cortex via the axon collaterals of these cells. Axon collateral feed back is thus already shown to be part of the cortical mechanism. The length of the period of inhibition of the cortical touch cells requires a long lasting series of inhibitory discharge. Such a discharge is produced by the Renshaw cells of the spinal cord and may well be present in certain cortical interneurones.

The work of CHANG (1950, 1951) demonstrating the existence of reverberating cortico-thalamic circuits, suggests as an alternative possibility, that the negative feed back may be mediated via these circuits. A latency of about 5 msec, which was observed for the inhibition of the touch cells, may be long enough to permit activation of an inhibitory cortico-thalamo-cortical path provided only a small number of interneurones are involved.

There are, however, several indications that something other than a simple negative feedback is involved in the delayed inhibition of the cortical touch cell. It has been seen that the initial discharge consists only of one or two impulses and is followed

by a long period of inhibition, whereas during the after-discharges the cell may fire 10 or 20 times at frequencies up to 800 per sec and not inhibit itself during this burst. If only a negative feedback were involved, one would expect that inhibition should increase in parallel with the frequency of discharge from the cell and thus cause a gradual shortening of the number of spikes per burst rather than the observed lengthening. Another observation contrary to the negative feedback concept is the almost complete inhibition of the cortical touch cell resulting from relatively low frequency stimulation of the afferent path. A series of afferent volleys at a frequency of 25 per second can stop the delayed discharge of the cell even though the cell may fire at much higher rates when the after discharge does occur and not inhibit itself. This again cannot be explained by a simple negative feedback system.

A mechanism which would explain the inhibition without invoking a negative feedback could be based upon a fractionation of the afferent inflow into a fast excitatory path and a slow inhibitory channel. A fractionation of the afferent inflow in the brain stem has been demonstrated by STARZL, TAYLOR and MAGOUN (1951). These workers have shown that in addition to the classical path of the specific afferents, a portion of the sensory inflow termed the "collateral afferents" plays into the brain stem reticular formation and exerts an influence upon the cerebral cortex some time after impulses from the specific conduction pathways have arrived at the cortex. The influence exerted by this collateral afferent system upon cortical activity seems to be largely of an excitatory nature mediated through the ascending reticular activating system (STARZL, et al., 1951). The descending influence of the reticular system is, however, known to cause inhibitory effects in the cord (MAGOUN, 1950), and LI (1956 b) has recently observed that the discharge of one thalamic nucleus, the nucleus ventralis lateralis, exerts an inhibitory effect upon the activity of the cortical motor neurones that may last from 80—400 msec.

The delayed cortical discharge of long duration and high frequency following the period of depression in a cortical cell seems also to depend on interaction between cortex and subcortical structures. A periodic variation of the excitability in cortical neurones was demonstrated in the auditory cortex by CHANG (1950, 1951). The primary cortical response was followed by a

series of depressions lasting approximately 50 msec and alternating with periods of facilitation lasting for 100 msec. The facilitation was shown due to impulses in reverberating cortico-thalamic circuits. LI, CULLEN and JASPER (1956) and LI (1956 a) have shown that a major influence of the diffuse thalamic projection system upon cortical cell activity is to increase the number of times a cell may fire in response to a given volley arriving via the specific thalamic relay nuclei. Activity of the diffuse thalamic nuclei may facilitate the cortical cell without causing it to fire. The nature and time course of the diffuse thalamic effect strongly suggest that this system may contribute to the delayed bursts demonstrated in the cortical touch cell response pattern.

Summary

1. Potentials evoked by stimulation of the chorda tympani and trigeminal component of the lingual nerve were studied by recording from the cerebral cortex of the cat. Single cortical cells influenced by these afferent paths were also investigated using KCl filled glass pipette microelectrodes.

2. Both the chorda tympani and the trigeminal component of the lingual nerve project to essentially the same cortical region on the orbital surface of the brain. A surface positive-negative wave is evoked by electrical stimulation of either path and this changes to a negative-positive wave at a depth of 0.5—0.8 mm below the cortex surface.

3. Single cortical cells were observed which responded to electrical stimulation of both the chorda and trigeminal paths; other cells responded only to stimulation of the trigeminal component of the lingual nerve, and a very few cells were seen which responded only to stimulation of the chorda tympani.

4. The initial component of the slow potential evoked by electrical stimulation of either the chorda or the trigeminal path was shown to result from activity in pathways activated by tactile stimulation of the tongue.

5. The receptive field of 34 cortical cells responding to tactile stimulation of the tongue was determined. Certain single cortical cells responded to stimulation of large areas of tongue surface, frequently from ipsilateral and contralateral sides. Other cells responded to a restricted area of a few square millimeters. No cortical touch cells responded to gustatory or thermal stimulation of the tongue.

6. Five cells were found responding only to gustatory stimulation of the tongue. These cells responded to almost all types of gustatory stimulation and lacked the specificity of response known for the chemoreceptors of the tongue. The cortical taste cells did not respond to any other sensory modality.

7. Several cortical cells were observed which responded only to thermal stimulation of the tongue.

8. The cortical touch cells fired in a definite pattern which was the same whether the afferent path was activated electrically or physiologically. The cell fired once or twice on the rising phase of a deep negative potential. This was followed by a period of depressed activity accompanied by a slow positive wave lasting 40—60 msec and then the cell fired again with bursts of impulses at high frequency alternating with pauses in the activity. The sequence of afterdischarges may last from 100 to 800 msec.

9. The period of depression after the initial discharge lasted as long as 150 msec and was shown due to an inhibitory influence exerted directly on the cortical cell. The long period of depression was not due to the refractory period of the cell as these cells were observed to fire at frequencies greater than 600 per sec.

10. The possible inhibitory mechanism is discussed and the physiological implications of the cortical touch cell discharge pattern are considered with reference to interaction between various cortical and subcortical regions.

References.

Albe-Fessard, D. and P. Busser, J. Physiol. Paris 1955. *47*. 67.

Amassian, V. E., EEG Clin. Neurophysiol. 1953. *5*. 415.

Benjamin, R. M. and C. Pfaffmann, J. Neurophysiol. 1955. *18*. 56.

Bremer, F., Arch. Int. Physiol. 1923. *21*. 308.

Brock, L. G., J. S. Coombs and J. C. Eccles, J. Physiol. 1952. *117*. 431.

Burns, B. D. and B. Grafstein, J. Physiol. 1952. *118*. 412.

Chang, H.-T., J. Neurophysiol. 1950. *13*. 235.

— Ibidem 1951. *14*. 95.

— Ibidem 1955. *18*. 452.

Chang, H.-T. and B. Kaada, J. Neurophysiol. 1950. *13*. 305.

Clare, M. H. and G. H. Bishop, EEG Clin. Neurophysiol. 1955. *7*. 85.

Cohen, M. J., S. Hagiwara and Y. Zotterman, Acta Physiol. Scand. 1955. *33*. 316.

Cragg, B. G., J. Physiol. 1954. *124*. 254.

Creutzfeldt, O., G. Baumgartner and L. Schoen, Arch. f. Psychiatr. u. Z. Neur. 1956. *194*. 592.

CURTIS, H. J., J. Neurophysiol. 1940. *3.* 414.
ECCLES, J. C., EEG Clin. Neurophysiol. 1951. *3.* 449.
ECCLES, J. C., P. FATT and K. KOKETSU, J. Physiol. 1954. *126.* 524.
ECCLES, J. C., P. FATT, S. LANDGREN and G. J. WINSBURY, J. Physiol. 1954. *125.* 590.
EYZAGUIRRE, C. and S. W. KUFFLER, J. Gen. Physiol. 1955 a. *39.* 87.
— Ibidem 1955 b. *39.* 155.
FATT, P. and B. KATZ, J. Physiol. 1953. *121.* 374.
FORBES, A. and B. R. MORISON, J. Neurophysiol. 1939. *2.* 112.
FRANK, K. and M. G. F. FUORTES, J. Physiol. 1955. *130.* 625.
GAD, A. P., Arch. Anat. Physiol. 1891. *15.* 541.
GEREBTZOFF, M. A., Arch. Int. Physiol. 1941. *51.* 199.
GRANIT, R., Receptors and Sensory Perception, 1955, Yale Univ. Press, New Haven.
HARRIS, W., British Med. J. 1952. *1.* 831.
JASPER, H. H. and C. AJMONE-MARSAN, Res. Publ. Ass. nerv. ment. Dis. 1950. *30.* 493.
KING, E. E., B. MINZ and K. R. UNNA, J. comp. Neurol. 1955. *102.* 565.
LELE, P. P., D. C. SINCLAIR and G. WEDELL, J. Physiol. 1954. *123.* 187.
LI, C.-L., J. Physiol. 1956 a. *131.* 115.
— Ibidem 1956 b. *133.* 40.
LI, C.-L. and H. JASPER, J. Physiol. 1953. *121.* 117.
LI, C.-L., C. CULLEN and H. JASPER, J. Neurophysiol. 1956. *19.* 111.
LILJESTRAND, G. and Y. ZOTTERMAN, Acta Physiol. Scand. 1954. *32.* 291.
MAGOUN, H., Physiol. Rev. 1950. *30.* 459.
PATTON, H. D. and V. E. AMASSIAN, J. Neurophysiol. 1952. *15.* 243.
PFAFFMANN, C., J. cell. comp. Physiol. 1941. *17.* 243.
PHILLIPS, C. G., Quart. J. exp. Physiol. 1956. *41.* 58.
RENSHAW, B., A. FORBES and B. R. MORISON, J. Neurophysiol. 1940. *3.* 74.
STARZL, T. E., C. TAYLOR and H. MAGOUN, J. Neurophysiol. 1951. *14.* 479.
TASAKI, I., H. POLLEY and F. ORREGO, J. Neurophysiol. 1954. *17.* 454.
ZOTTERMAN, Y., Skand. Arch. Physiol. 1935. *72.* 73.
— Ibidem 1936. *75.* 105.
— Acta Physiol. Scand. 1949. *18.* 141.

524.
ysiol.
. 87.
ress,
ient.
102.
187.
1.
954.
243.
940.
951.
17.

STOCKHOLM 1957. KUNGL. BOKTRYCKERIET P. A. NORSTEDT & SÖNER